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Perianal verrucous epidermal naevus mimicking perianal warts

A case of perianal verrucous epidermal naevus mimicking perianal warts in a 2 year old boy is described. Verrucous epidermal naevus should be included in the differential diagnosis of perianal warty lesions, particularly when they are present since birth or appear during childhood.

CASE REPORT

A 2 year old boy was referred by a paediatrician for the evaluation of a perianal verrucous lesion which looked like perianal warts. The condition was first noticed by the child's mother when he was 9 months old as a raised velvety area around the anal orifice. Over the next few months, multiple, small, warty elevations developed over the region. The lesions had remained stable thereafter. There was no parental report of scratching, exudations or bleeding, or difficulty in passing stools. There was no history of viral warts or any STD in the parents. The child has remained in good health since his birth and achieved the milestones normally. Examination revealed a mildly elevated, velvety, periorificial skin studded with multiple, brownish, keratotic papules (fig 1).

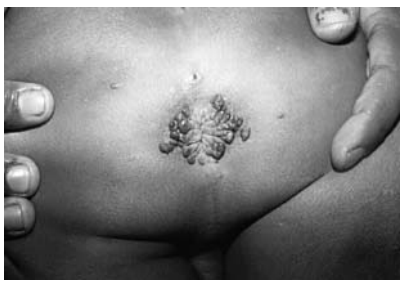

Figure 1 Perianal warty papules.

Detailed systemic examination failed to reveal any abnormality.

A provisional diagnosis of verrucous epidermal naevus was made and a punch biopsy specimen was obtained. Histological examination corroborated the clinical diagnosis by showing hyperkeratosis, acanthosis, and papillomatosis without any evidence of vacuolar change in the keratinocytes or any dermal pathology. Virological study for human papillomavirus (HPV) could not be done owing to lack of facilities. The parents declined any immediate treatment for the asymptomatic condition and during a follow up period of 1 year, the child has remained healthy with the lesions remaining unchanged in appearance.

COMMENT

Verrucous epidermal naevi are circumscribed hamartomatous lesions composed almost exclusively of keratinocytes.[1] Most epidermal naevi usually occur at birth or infancy but rarely their appearance may be delayed until puberty.[2] The lesions typically consist of closely set warty papules that coalesce to form well defined keratotic plaques usually in a linear fashion. Verrucous epidermal naevi may be almost of any size, may be single or multiple, and can occur at more or less any site.[1] Since these lesions closely mimic viral warts, their occurrence in the perianal region during childhood or adolescence may raise the suspicion of perianal warts as in the present case. Onset of the lesions early in life, their stable nature, typical linear configuration, and histological features may help in the differential diagnosis. Usually only of cosmetic importance, the skin lesions may be treated by cryotherapy, surgical excision, or carbon dioxide laser ablation.[1 3]

Epidermal naevi, particularly if extensive, may be associated with other developmental anomalies mainly involving the central nervous system, the skeletal system, and the eyes.[4] In a large study, one or more such abnormalities were demonstrated in 33% of cases.[2] Since patients with epidermal naevi are at significant risk of having other abnormalities, detailed systemic examination and periodic follow up is warranted in every case to exclude them.

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Investigating the microbial aetiology of pelvic inflammatory disease

An effort to elucidate a subject which is laden with difficulties is noteworthy, so that it was interesting to read the report by Simms *et al*[1] on the associations between *Mycoplasma genitalium*, *Chlamydia trachomatis*, and pelvic inflammatory disease (PID). The difficulties are at least threefold. Firstly, a diagnosis of PID based on symptoms and clinical signs, as in the study reported, is acknowledged, both generally and by the authors, to be imprecise. Clinical observations often do not tally with laparoscopic findings,[2] laparoscopy being a fundamental diagnostic requirement in research investigations. Secondly, it is obvious that specimens cannot be taken from the inflamed site in question without laparoscopy. Indeed, it is axiomatic that this should be done if there is to be any chance of unravelling the microbial aetiology. Taking specimens from the cervix is very much second best as the results of microbiological testing may bear no relation to the pathological changes in the tubes. Thirdly, and no less relevant, is the question of an adequate control group. It seems that this should not comprise women undergoing tubal ligation. Although a source of normal tubes would seem sensible, the women were not in the same cohort as those with disease and, in any event, for comparative purposes specimens were taken from the cervix. Surely, an examination of specimens from women without symptoms and signs of PID but who were otherwise comparable to those who did have symptoms and signs would have been more appropriate? In future investigations, controls should be women within a laparoscopically based study who are found not to have PID on laparoscopy. Even then, the situation may be clouded because, in one study,[3] *C trachomatis* was detected as often in the tubes of women who did not have PID visually as in those of women who did. Certainly, however, finding *M genitalium* in the cervix of women with ill defined PID significantly more often than in the cervix of women who did not have PID and who, in other ways, appeared not to be comparable may mean nothing in relating *M genitalium* to tubal pathology. It is a far cry from unravelling the role of *M genitalium* in PID, despite some strong suggestions that it might be involved.[4]

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Lack of evidence for sexual transmission of hepatitis C virus in patients attending STD clinics in Pune, India

The presence of hepatitis C virus (HCV) RNA in semen among two of six (33%) HIV negative and six of 15 (40%) HIV infected males, reported recently suggests that HIV may facilitate genital shedding and subsequent sexual transmission of HCV.[1] We determined HCV prevalence and examined evidence for its sexual transmission in a cohort of STD patients with observed HIV prevalence of 21.2%.

Consecutive serum samples (n = 9141) collected between January 1994 and December 1999 were batched, pooled, and tested for anti-HCV antibody (Ortho HCV 3.0, Ortho-clinical Diagnostic, Germany). As previously described,[2] 25 µl aliquots of five samples were pooled and 20 µl of each pool were screened. Samples from positive pools were then tested individually. Positive sera were tested by HCV RNA polymerase chain reaction (PCR) using standard primers.[3] HIV antibody status of each sample was ascertained using the algorithm described previously.[4] Data were analysed using statistical package SPSS version 10.0. This study was a part of a prospective cohort study that was approved by ethics committee/institutional review boards of the collaborating organisations and blood samples were collected after counselling and informed consent.

Overall prevalence of anti-HCV antibodies was 0.68% (62/9141, 95% CI 0.52 to 0.87). The prevalence among HIV infected individuals (1.5%, 95% CI 1.0 to 2.1) was higher (p = <0.01) than that in those not infected (0.44%, 95% CI 0.3 to 0.6). The annual anti-HCV antibody prevalence rate between 1994 and 1999 was 0.57%, 0.46%, 1.10%, 0.81%, 0.37%, and 0.61%, which did not change significantly over time (table 1). Of the 55 anti-HCV antibody positive sera tested, 27 (49%) were HCV RNA PCR positive.

Univariate analysis revealed that history of past or current STD was not associated with HCV, whereas female sex (OR = 2.07, 95% CI 1.17 to 3.66), prevalent HIV infection (OR = 3.38, 95% CI 2.05 to 5.58), history of tattoo (OR = 2.18, 95% CI 1.31 to 3.63), and being a sex worker (OR = 2.35, 95% CI 1.27 to 4.35) were significantly associated with presence of anti-HCV antibody. However, multivariate analysis revealed that prevalent HIV infection and tattooing increased the likelihood of presence of anti-HCV antibodies by 3.08-fold (AOR 3.08, 95% CI 1.86 to 5.11, p = <0.00) and 1.87-fold (AOR 1.87, 95% CI 1.12 to 3.13, p = 0.017), respectively (table 1).

Table 1 Characteristics of study participants and association with prevalent anti-HCV antibody

Variable	No	Anti-HCV antibody positive (%)	Unadjusted OR (95% CI)	p Value	Adjusted OR (95% CI)*	p Value*
1 Year screened					Not included in multivariate analysis	
1994	1901	11 (0.57)	1 (Referent)			
1995	1933	9 (0.46)	0.80 (0.33 to 1.94)	0.628		
1996	1997	22 (1.10)	1.91 (0.93 to 3.96)	0.08		
1997	1109	9 (0.81)	1.41 (0.58 to 3.40)	0.45		
1998	1064	4 (0.37)	0.65 (0.21 to 2.04)	0.459		
1999	1135	7 (0.61)	1.07 (0.41 to 2.76)	0.895		
TOTAL	9139	62 (0.67)				
2 Males who had contact with sex worker					Not included in multivariate analysis	
YES	6281	40 (0.69)	1.63 (0.69 to 3.86)	0.259		
NO	1535	6 (0.39)	1 (Referent)			
TOTAL	7816	46 (0.58)				
3 Sex						
Women	1323	16 (1.21)	2.07(1.17 to 3.66)	0.013		0.469
Men	7816	46 (0.59)	1 (Referent)			
Total	9139	62 (0.67)				
4 Sex worker						
Yes	933	13 (1.39)	2.35 (1.27 to 4.35)	0.006		0.231
No	8206	49 (0.59)	1 (Referent)			
Total	9139	62 (0.67)				
5 HIV serostatus						
Pos	2102	31 (1.47)	3.38 (2.05 to 5.58)	<0.001	3.08 (1.86 to 5.11)	<0.001
Neg	7037	31 (0.44)	1 (Referent)		1 (Referent)	
Total	9139	62 (0.67)				
6 History of tattoo						
Yes	3703	37 (0.98)	2.18 (1.31 to 3.63)	0.003	1.87 (1.12 to 3.13)	0.017
No	5424	25 (0.46)	1 (Referent)		1 (Referent)	
Total	9127	62 (0.67)				

*Multivariate analysis was done using binary logistic regression by forward LR method.
OR = odds ratio.

A rapid spread and high HCV prevalence of 80% has been reported recently among a cohort of injecting drug users from Kolkata, India.[5] In contrast, we observed a low and stable prevalence of anti-HCV antibody among STD clinic attendees over the past 6 years in an urban setting where HIV transmission was predominantly sexual. Given that a high HIV prevalence was reported among female sex workers (FSWs) in this population[4] and about 70% of males attending STD clinic had visited FSWs in the past 3 months, stable HCV prevalence over 6 years suggests that HCV is not efficiently transmitted sexually. Additionally, no association was found between past or current STD and HCV prevalence, and a high prevalence and incidence of HBV, a known sexually transmitted infection, have been reported in this population.[6] Our analysis failed to identify any evidence that could support sexual transmission of HCV.

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Monosymptomatic hypochondriacal psychosis

Dr O'Mahony illustrates in his literary and graphic way the difficulties associated with dealing with this condition (from which his patient was almost certainly suffering).[1] It is good to know that his hospital is taking seriously the issue of actual or threatened violence to staff. Having had several similar cases over the past couple of years, including one who eventually committed suicide, I have been able to make appropriate arrangements with a psychiatrist who was unequivocal in his advice that he should be in on a subsequent consultation right from the start and be introduced to the patient as a double consultation. The ethics of this include the fact that such delusional patients are, of course, psychotic and unable to bring rational decision making processes to the problem.